Fragments of Ang-1, lacking a significant portion of the N-terminus of Ang-1 are also preferred. Desirably, such truncated Ang-1 peptide portions comprise less than about 50%, more preferably less than about 60%, of the Ang-1 amino acid sequence. Preferably, the Ang-1 truncated peptide portion is truncated in the N-terminal portion of the Ang-1 amino acid sequence. Truncated Ang-1 peptide portions lacking all or part of the predicted Ang-1 alpha helix rich coiled coil domain (SEQ ID NO: 18) (e.g., at least 10%, preferably at least about 50%, and more preferably at least about 90% of either the C-terminus or N-terminus of the domain, or both) are also desirable (other predicted coiled coil domains, including possible Ang-1 coiled coil domains are discussed further herein), as are Ang-1 peptide portions lacking the variable N-terminal domain (SEQ ID NO: 19) (similar modifications can be applied to other angiopoietin peptide portions and angiopoietin related factor peptide portions). Fusion proteins including such truncated Ang-1, or, more preferably, KAP peptide portions, may permit better binding to the KDR and TIE-2 receptors. Fusion proteins that exhibit higher affinity for both the KDR and TIE-2 receptors over full length VEGF-Ang-1 homologs are preferred. Moreover, due to the non-heparin binding nature of the preferred VEGF peptide portion, binding with undesired receptors (e.g., neuropilin-1) is reduced, thereby increasing TIE-2/KDR interaction.

A2

Replace paragraph [0083] with:

As indicated above, in some contexts a fusion protein consisting of a heparin-binding VEGF peptide portion is preferred over fusion proteins comprising a non-heparin-binding VEGF peptide portion. Accordingly, such fusion proteins also are provided by the invention. In general, the principles applicable to the non-heparin-binding VEGF peptide portion are also applicable to such heparin-binding VEGF peptide portions, except with respect to factors such as mobility (discussed with respect to non-heparin-binding VEGF fusion proteins below), pH (as discussed above), and/or protein interactions (e.g., neuropilin interactions or VEGF receptor interactions), which typically will vary from those described above with respect to non-heparin-binding VEGF peptide portions (i.e., by exhibiting biological activity similar to heparin-binding VEGFs, such as $VEGF_{189}$ or $VEGF_{165}$). The heparin-binding VEGF peptide portion can comprise any suitable heparin-binding VEGF (e.g., a $VEGF_{189}$ or homolog thereof). $VEGF_{165}$, heparin-binding fragments thereof, and homologs thereof, are preferred wild-type and wild-type-derived heparin binding VEGF peptide portions components. Other advantageous heparin-binding VEGFs include VEGFs derived from $VEGF_{121}$, which typically generated through addition of the heparin-binding domain of another VEGF, such as $VEGF_{189}$ or an artificial heparin-binding domain. Examples of such VEGFs include $VEGF_{121.2}$ (SEQ ID NO: 60) and $VEGF_{121.3}$ (SEQ ID NO: 61), which include

A3